

Toward Evidence-Based Early Interventions for Acutely Traumatized Adults and Children

Introduction

Terrorism is fundamentally psychological warfare. The current threat of chemical/biological/nuclear attacks causing massive casualties is real. The present climate of fear and uncertainty is a global problem of the highest priority that demands our immediate attention.

Very shortly after the September 11 terrorist attacks, civil leaders and policy makers turned to the mental health community for guidance. "What evidence-based interventions work best in the acute aftermath of such a devastating catastrophe?" they asked. "Given the massive number of people affected in New York City alone, to say nothing of multitudes of distressed families, friends and onlookers in surrounding areas, how can we distinguish those at greatest risk from those who will recover on their own? And what about the children? How can we help them?"

Unfortunately, there are no clear answers to these questions. Although a wealth of information had been accumulating in recent years concerning adaptive and pathologic responses to traumatic stress, only a handful of rigorous studies had tested acute psychosocial and pharmacological interventions. This is hardly a sufficient clinical database on which to justify public mental health policy decisions affecting millions of people and costing hundreds of millions of dollars.

Responding to the urgent needs of our society, in June 2002, the Anxiety Disorders Association of America (ADAA) convened a conference to address key conceptual models and scientific findings pertinent to the phenomenology, psychology, psychobiology, and evidence-based early interventions for adults and children acutely exposed to catastrophic events. This conference was sponsored by ADAA, the National Institutes of Mental Health (NIMH), and the Department of Veterans Affairs National Center for Posttraumatic Stress Disorder (PTSD). It was supported by an unrestricted educational grant from the Eli Lilly Pharmaceutical Company. Many of the papers presented at this conference are included in this special issue of *Biological Psychiatry*.

Above and beyond topics addressed in specific papers, the overriding scientific and public health questions will translate our current scientific knowledge into evidence-based practices and feasible public policies that will foster resilience, promote preventive strategies, and provide effective early intervention. It is crucial to explicate the most important research questions that address the current

crisis. In short, what do we know, what don't we know, and what should we do to counteract the effects of traumatic experiences? We'll return to these questions after a brief introduction to the articles in this special volume.

Phenomenology

The panel on phenomenology considered normal and pathologic responses to acute trauma, factors that predict chronicity, and concerns with respect to children and adolescents.

Norris et al (2003), after presenting epidemiologic results on over 2500 adults from four Mexican communities, report that the most common posttraumatic reaction was mild (present but below PTSD symptom criteria), immediate (within the first month), and transient (over within a year). Although 29% experienced reactions that were immediate and serious of whom, almost half (44%) developed chronic PTSD symptoms. In addition, serious/immediate chronic reactors were distinguished from serious/immediate transient reactors by a greater level of functional impairment, depressive and somatic symptoms. These findings have some important implications about the best way to set priorities for mental health triage following mass casualties.

It is reasonable to ask whether there is any constellation of acute posttraumatic symptoms that predicts the later development of PTSD or other psychiatric disorders. Bryant (2003) reviews the literature concerning the predictive power of Acute Stress Disorder (ASD), which became an official diagnosis with little empirical support. He shows that whereas a significant proportion of people meeting ASD criteria go on to develop PTSD, 29%–72% of people who develop PTSD have never met ASD criteria. In other words, the majority of people at risk for PTSD may never be detected in advance if screening is restricted to the presence or absence of ASD. Bryant goes on to review other acute stress symptoms that may be better predictors of PTSD than ASD. He concludes that there "are multiple pathways to PTSD development" and that "there may be greater utility in focusing on the interaction between symptoms, biological responses and cognitive factors in predicting who will develop PTSD."

March (2003) reports that there is little empirical evidence that ASD occurs in children and adolescents or that ASD is a good predictor of the subsequent develop-

ment of PTSD. He also points out that research with acutely traumatized children is limited by cross-sectional designs and has not paid sufficient attention to developmental factors, parental stress, or specific stressor characteristics. He presents a testable, multivariate model that is developmentally sensitive in which putative moderating and mediating variables and processes are identified.

Psychological and Biological Mechanisms

This segment of the ADAA conference reviewed conceptual models that can inform our research on acute psychological and biological responses to traumatic stress, their prevention and treatment.

McNally (2003) reviews cross-sectional research on psychological mechanisms suggesting that social support, intelligence, neurologic soft signs, and neuroticism may all be associated with vulnerability to develop PTSD. A more conceptually rich approach is to consider peritraumatic factors that may predict PTSD such as: peritraumatic dissociation, peritraumatic threat appraisal (i.e., the belief one is about to die) and negative self-relevant appraisal (i.e., shame, guilt, or sense of incompetence). Finally, McNally suggests how there may be differences in adaptive versus pathologic cognitive operations by which traumatic information is processed initially and encoded as memories.

Pine's (2003) review on developmental psychobiological and behavioral responses to threat is best understood within the context of animal research on fear conditioning. Indeed the neural circuitry and neurobiological systems exhibiting alterations in PTSD are those that would have been expected to show such abnormalities based on laboratory research findings. With regard to children, Pine cites several studies suggesting that exposure to stress during crucial developmental periods may produce "long-term, potentially semi-permanent alterations in stress response systems."

Early Intervention

The final segment of the ADAA conference focused on the evidence regarding the effectiveness of psychosocial and pharmacological early interventions for adults and children.

Ehlers and Clark (2003) review the current literature on psychological debriefing, a very popular early intervention, and cognitive-behavioral therapy (CBT), a powerful treatment for chronic PTSD that has been utilized as an early intervention to facilitate recovery in a few studies. In short, single session psychological debriefing does not prevent PTSD and may actually retard recovery. Brief CBT protocols, on the other hand, have been shown to

effectively reduce the subsequent development of PTSD and to ameliorate ASD symptom severity. This article also cites several psychological mechanisms that may predict PTSD following traumatic exposure

Morgan et al (2003) provide a review of psychobiological mechanisms underlying the human stress response that focuses on the balance between excitatory (i.e., glutamatergic), inhibitory (i.e., GABA-ergic) and modulator (i.e., peptides, monoamines and hormones) brain mechanisms. Their review provides an excellent context within which to consider conceptually driven pharmacological approaches to early intervention.

Finally, Cohen (2003) provides a thoughtful summary of the pediatric literature. In short, three randomized clinical trials, along with several open studies, suggest that CBT is also effective in children as it is in adults. The optimal timing of such interventions has not been studied systematically, but as with Ehlers and Clark, there is evidence to suggest that better outcomes may be achieved if treatment is delayed for several months rather offered shortly after acute traumatization. The few pharmacological studies published thus far suggest that imipramine and opiates may protect against the later development of PTSD in traumatized children.

What do we know? What don't we know? What should we do?

Phenomenology

WE KNOW. The general response to trauma is one of immediate and significant distress; most people will recover spontaneously, but a sizable minority will progress to a chronic incapacitating disorder such as PTSD or depression; it is very difficult to predict who will recover and who will go on to develop trauma-related chronic disorders; Acute Stress Disorder has limited usefulness as a screening criterion because most people who develop PTSD never meet diagnostic criteria for ASD; and very little current scientific information pertains to children.

WE DON'T KNOW. What is the full range of psychological reactions in the acute phase with respect to both symptom profiles and functional impairment; how do we take into account age, gender, and cultural differences; what measurable acute posttraumatic phenomenological, diagnostic, psychological, and biological factors will permit us to distinguish resilient survivors from those vulnerable to develop PTSD and other psychiatric problems; and what are the best methods and instruments for evaluating posttraumatic distress and for monitoring affected individuals over time?

WHAT WE SHOULD DO. We can close the gaps in knowledge by developing models that characterize the full

range of acute phase reactions and predict chronicity; conducting epidemiologic research on the general population and longitudinal studies on specific vulnerable/resilient groups; developing standard and reliable instrumentation and procedures for these research activities; and promoting separate initiatives for children.

Psychological and Biological Mechanisms

WE KNOW. A number of psychological mechanisms such as negative cognitions threat appraisal, coping behaviors, information/memory processing, and cognitive strategies predict PTSD; animal models of fear conditioning and neurobiological models concerning the human stress response provide a useful context for understanding acute distress and PTSD; and exposure to stress during crucial developmental periods may produce stable abnormalities in stress response systems.

WE DON'T KNOW. What psychological and biological reactions constitute an adaptive human response to traumatic stress; how do we distinguish adaptive from maladaptive responses or when to do so; what psychological and biological mechanisms are involved in normal recovery from traumatic stress; and what may be unique about children in this regard?

WHAT WE SHOULD DO. We can close the gap in knowledge by conducting longitudinal studies with high risk populations in which psychological and biological variables are monitored which appear to predict vulnerability and resilience; promoting laboratory research assessing the relationship between clinical symptoms and specific psychological and biological mechanisms; promoting intervention research in which protocols and potential psychological and biological change mechanisms are monitored along with clinical outcomes; and extending such research approaches to traumatized children at different developmental stages.

Early Intervention

WE KNOW. Randomized clinical trials with CBT interventions have been successful in accelerating recovery and/or reducing PTSD incidence; randomized trials on individual psychological debriefing indicate that this popular early intervention is either ineffective or may actually delay recovery; acute pharmacotherapeutic interventions have been tested very sparingly; and there are no empirical studies on acute psychosocial interventions for children.

WE DON'T KNOW. How can knowledge of psychological and biological mechanisms be translated to effective treatments; what treatments at what times will be helpful to trauma survivors; what acute psychotherapeutic or pharmacological interventions can be recommended at this

time; and what societal interventions such as education, preventive actions, community interventions, and risk communication strategies should be recommended at this time?

WHAT WE SHOULD DO. We can close the gap in knowledge by investigating a wide spectrum of individual, group, and community interventions. Research on individual interventions should consider efficacy, effectiveness, timing, treatment setting, dosage, target population, cultural factors, and developmental level. Research on group interventions should rigorously test group debriefings, self-help initiatives, as well as other psychosocial approaches. Research on societal and community level interventions should systematically evaluate: pretraumatic preparation as well as posttraumatic community/societal interventions and evaluate a range of outcomes at both the individual and community level such as: adaptive functioning, mental/physical health, knowledge/attitudes concerning trauma, effective coping and health-seeking behavior.

Conclusion

The challenge is urgent and of the highest importance. Given the complexity of the issues, paucity of data, and lack of scientific evidence to guide public policy, much research needs to be done. The basic challenge is to develop psychological and biological models that distinguish normal recovery from pathological reactions, resilience from vulnerability, and conceptually driven interventions that work. Separate initiatives must be undertaken for children because of special cognitive, neurobiological, developmental, and parental issues that must be considered.

We consider the ADAA conference, itself, an early intervention for the scientific and mental health community. Hopefully, it will promote translational research that will help us become wiser and better prepared to respond to any mass casualty in the future.

Matthew J. Friedman

National Center for PTSD (116D)
215 N. Main Street
VA Medical and Regional Office Center
White River Junction, VT 05009

Edna B. Foa

Department of Psychiatry
University of Pennsylvania
Philadelphia, Pennsylvania

Dennis S. Charney

Mood and Anxiety Disorders Program
National Institute of Mental Health
Bethesda, Maryland

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